Laboratory Sleep Correlates of Nightmare Complaint in PTSD Inpatients

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Background: *Nightmares are rare in the sleep laboratory, even in patients with posttraumatic stress disorder for whom nightmare complaints are diagnostic. Nevertheless, it is possible that laboratory conditions do not preclude the observation of telltales—nightmare-related modifications of tonic sleep—given sufficiently large samples.*

Methods: *Sixty-three unmedicated, nonapneic Vietnam combat veterans undergoing inpatient treatment for posttraumatic stress disorder underwent polysomnographic testing and assessment of nightmare complaint.*

Results: *Trauma-related nightmare complaint, but not non–trauma-related complaint, was associated with increased wake-after-sleep-onset in the sleep laboratory. No relationships between nightmare complaint and rapid eye movement sleep architecture were observed.*

Conclusions: *Increased wake-after-sleep-onset was specifically associated with trauma-related nightmare complaint, confirming data from other quarters suggesting they are both phenomenologically and functionally distinct from normal dreaming.* Biol Psychiatry 2000;48: 1081–1087 © *2000 Society of Biological Psychiatry*

Key Words: Sleep, dreams, anxiety, stress, traumatic

Introduction

The sleep researcher studying posttraumatic stress disorder (PTSD) must confront, in one fashion or another, the fact that the most salient and specific sleep disturbances of this disease, trauma-related nightmares (TRNs), are not reliably observed in the sleep laboratory. The literature contains 12 laboratory studies of PTSD patients in which nightmare base rates can be estimated with some confidence. Four early studies observed nightmares on at least 10% of nights in the laboratory (Hefez et al 1987; Lavie et al 1979; Schlosberg and Benjamin 1978; van der Kolk et al 1984)[1]; however, more recent studies reporting on a much larger aggregate sample have observed nightmares on less than one percent of laboratory nights (Dow et al 1996; Glaubman et al 1990; Mellman et al 1995a, 1995b, 1997; Ross et al 1994; Woodward et al 1996a, 1996b). It is not obvious why these two sets of studies have had different rates of TRNs in the laboratory, though it should be noted that the earlier studies tested patients sooner after trauma. If nightmare frequency was judged from the latter and larger set, it is unclear whether they would merit inclusion among the diagnostic criteria for PTSD. Nevertheless, the existence and significance of TRNs has not been seriously questioned.

Perhaps one reason a relaxed degree of skepticism has been accorded TRNs is that non–trauma-related nightmares (NTRNs) also occur at lower than reported rates in the laboratory. Fisher (Fisher et al 1970) suggested that the presence of another (awake) person in the laboratory, just outside the subject's sleeping quarters, might substantially reduce the likelihood of the nightmares. It is interesting to note that PTSD patients have also generally failed to demonstrate modifications of tonic sleep parameters in the laboratory that are commensurate with their complaints of impaired sleep initiation and maintenance. Contrary to the case of nightmares, this result has often led to skepticism regarding the existence of PTSD-related sleep maintenance disturbances. Although it is not inconceivable that nightmares are ameliorated in the sleep laboratory, whereas other features of the sleep of PTSD patients are not, there is little empirical support for this position. Alternatively, it is possible that most or all PTSD-related sleep changes are substantially ameliorated in the "guarded" context of the laboratory, and there fall prey, in a statistical sense, to the large underlying normative variation in sleep architecture. If the conditions of the sleep laboratory exert an ameliorative effect on sleep in PTSD, it remains possible that *telltales* of PTSD-related sleep disturbance may be discerned in the lab given adequate sample sizes and analytic methods.

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[1]The percentage of nights with nightmare reports across these four studies could rise to 41% if indeterminate numbers of nightmares and nights observed were set to their respective upper and lower bounds.

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Reported below are sleep architectural finding associated with the presence or absence of nightmare complaints in a sample of 63 Vietnam combat–related PTSD inpatients. Based upon the analysis of a large psychometric data set collected in our laboratory, these analyses will further distinguish between TRN complaint and NTRN complaint. The psychometric data will be briefly summarized. In a sample of 400 combat veterans admitted to the inpatient PTSD treatment program at the National Center for PTSD, Clinical Laboratory and Education Division, the distributions of TRN and NTRN nightmare complaints over patients were significantly different (Table 1). Furthermore, patients with TRN complaint reported significantly higher combat exposure than those without (Figure 1), whereas patients with and without NTRN complaint did not differ in combat exposure (cf. Neylan et al 1998). Finally, TRNs, but not NTRNs, were associated with elevations on scales 6 ("paranoia") and 8 ("schizophrenia") on the MMPI/MMPI-II (Butcher et al 1989; Figure 2). It was therefore of interest to determine whether patients with TRN and NTRN complaints—classified using the same sleep history questionnaire (Figure 3)—would exhibit differences in objective sleep.

Table 1. Distribution of Nightmare Complaint Vietnam Combat–Related PTSD Inpatients

	Non–trauma-related nightmares		
	–	+	Total
400 admissions to inpatient treatment program (in percentages)			
Trauma-related nightmares			
–	8.25	5.5	13.75
+	30.75	55.5	86.25
Total	39.0	61.0	100
Study sample			
Trauma-related nightmares			
–	10	5	15
+	14	34	48
Total	24	39	63

Cross-tabulations of trauma-related nightmare vs. non–trauma-related nightmare complaint in 400 posttraumatic stress disorder (PTSD) inpatients and the study sample. The distributions of complaints were significantly different over both [N = 400, χ^2 (1) = 15.7, p = .001; N = 63, (χ^2 (1) = 6.9, p = .009].

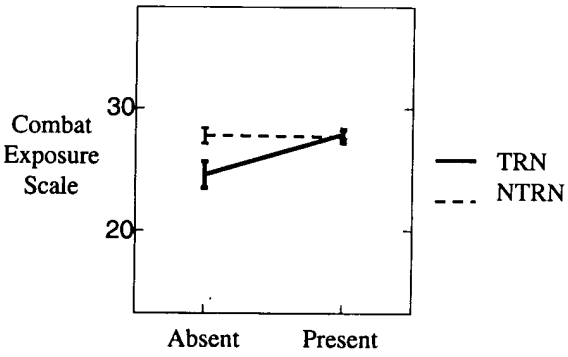

Figure 1. Combat Exposure Scale (CES) scores by nightmare type in 400 posttraumatic stress disorder (PTSD) inpatients. Subjects were nonconsecutive admissions to the same PTSD inpatient treatment unit from which the laboratory study sample was drawn. CES scores differed as a function of the presence vs. absence of trauma-related nightmares (TRNs) vs. non–trauma-related nightmares (NTRNs). TRN effect, $F(1,396) = 7.8$, $p = .005$; NTRN effect, $F(1,396) = 0.03$, ns; TRN × NTRN effect, $F < 1$.

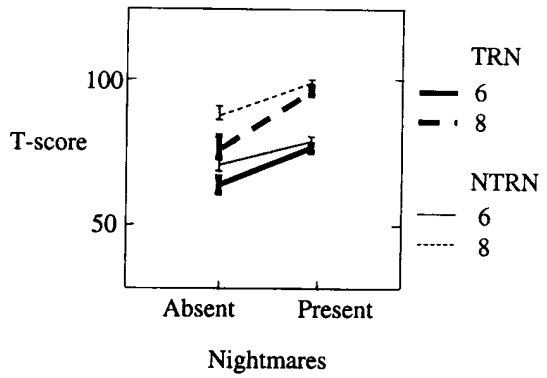

Figure 2. Minnesota Multiphasic Personality Inventory (MMPI) scales 6 and 8 T scores by nightmare type in 400 posttraumatic stress disorder (PTSD) inpatients. In the larger sample, MMPI scale 6 and 8 scores also differed as a function of the presence vs. absence of trauma-related nightmares (TRNs) vs. non–trauma-related nightmares (NTRNs). Scale 6 TRN effect, $F(1,395) = 10.3$, $p < .001$; scale 8 TRN effect, $F(1,395) = 19.1$, $p < .001$; scale 6 NTRN effect, $F(1,395) = 2.1$, $p = .1$; scale 8 NTRN effect, $F(1,395) = 1.4$, ns; TRN × NTRN effects, Fs < 1.

Methods and Materials

Subjects

Combat-related PTSD inpatient subjects were recruited from the Specialized Inpatient PTSD Unit at the Veterans Administration Medical Center, Palo Alto, California. Potential subjects with evidence of medical disease and/or chronic pain that could influence sleep were excluded, as were individuals with risk factors for obstructive sleep apnea (frequent snoring, obesity, or partner reports of interrupted breathing during sleep). Subjects were also excluded from analysis if, on any laboratory night, there was evidence of sleep apnea as indicated by an apnea/hypopnea index greater than 10 events per hour or a periodic limb movement arousal index of greater than 10 events per hour. Subjects were excluded if there was a recent history of heavy alcohol use (intake greater than 5 oz/day for any 30-consecutive-day period during the preceding 6 months). All included subjects reported having been abstinent from alcohol or illicit drugs for at

NAME: _____ SSN: _____ AGE: ____ EDUCATION: ____
(Please print.)
I USUALLY GO TO BED AT _____ .

I USUALLY GET OUT OF BED (FOR THE LAST TIME, OR "IN THE MORNING") AT _____ .

I WOULD RATE MY SLEEP AS (Circle one)... 1 EXCELLENT. 2 SATISFACTORY. 3 POOR. 4 VERY POOR.

IT USUALLY TAKES ME _____ MINUTES TO FALL ASLEEP AT NIGHT.

I USUALLY AWAKEN _____ TIMES DURING THE NIGHT, AT _____ , _____ , _____, _____.
(TIMES)

IF I AWAKEN , IT USUALLY TAKES ME _____ MINUTES TO FALL BACK ASLEEP.

I USUALLY NAP FOR _____ MINUTES DURING THE DAY.

I EXPERIENCE COMBAT NIGHTMARES...
1 RARELY OR NEVER.
2 ONCE OR TWICE PER MONTH.
3 ONCE OR TWICE PER WEEK.
4 ALMOST NIGHTLY.

I EXPERIENCE NON-VIETNAM NIGHTMARES OR UNPLEASANT DREAMS...
1 RARELY OR NEVER.
2 ONCE OR TWICE PER MONTH.
3 ONCE OR TWICE PER WEEK.
4 ALMOST NIGHTLY.

I EXPERIENCE PLEASANT DREAMS...
1 RARELY OR NEVER.
2 ONCE OR TWICE PER MONTH.
3 ONCE OR TWICE PER WEEK.
4 ALMOST NIGHTLY.

I FEAR GOING TO SLEEP... 1 VERY MUCH. 2 A LITTLE. 3 NOT AT ALL.

IN EACH BOX, PLACE A "Y" IF A RELATIVE HAD THAT SYMPTOM, "N" IF THEY DEFINITELY DID NOT, "?" IF YOU'RE NOT SURE, AND A "N/A" IF THE CATEGORY DOES NOT APPLY TO YOU .

	BIOLOGIC FATHER	BIOLOGIC MOTHER	FULL BROTHERS	FULL SISTERS	SPOUSE	BIOLOGIC CHILDREN	ADOPTED CHILDREN
SNORER							
NIGHTMARER							
SLEEPWALKER							
INSOMNIAC							
ALCOHOLIC							

WHEN I WAS A CHILD, I...
SLEEP-WALKED..... ___
HAD NIGHTMARES..... ___
OFTEN SLEPT WITH SIBLING OR PARENT..... ___
LIKED A LIGHT ON NEARBY..... ___
WAS A VICTIM OF NIGHT-TIME ABUSE..... ___

IN THE MILITARY, I EXPERIENCED. (HOW MANY?)
_____ NIGHTS OF NIGHT-COMBAT TRAINING
_____ NIGHTS SLEEPING IN THE BUSH
_____ NIGHT-TIME FIREFIGHTS, AMBUSHES, ETC.

I I HAVE SOUGHT PROFESSIONAL HELP REGARDING SLEEP DIFFICULTY..... ___
I HAVE TAKEN SLEEP MEDICATIONS..... ___

THINGS THAT IMPROVE MY SLEEP... _____
THINGS THAT WORSEN MY SLEEP... _____

SINCE VIETNAM, I HAVE HAD _____ AUTO ACCIDENTS AND _____ WORK-RELATED ACCIDENTS.

I HAVE EXPERIENCED _____ MONTHS HOMELESS, SLEEPING OUTSIDE, IN THE STREETS, OR IN SHELTERS.

I SNORE ..... _____ NEVER _____ SOMETIMES _____ USUALLY _____ ALWAYS

I HAVE BEEN TOLD THAT I.....
MOVE MY LEGS FREQUENTLY DURING THE NIGHT..... ____
HAVE VIOLENT NIGHTMARES DURING WHICH I OFTEN ATTACK MY PARTNER..... ____

Figure 3. Sleep history questionnaire.

least 30 days before hospitalization. In addition, all included subjects were free of prescription psychotropic medication on admission to the inpatient program and were maintained off medications before testing. Mean duration of hospitalization before testing was 35 days. Finally, no subject was medicated with a selective serotonin reuptake inhibitor within 80 days of testing.

The final sample consisted of 63 Vietnam combat–related PTSD inpatients meeting DSM-IV criteria for current PTSD as the primary diagnosis determined through administration of the Structured Clinical Interview for the DSM-III-R (SCID; American Psychiatric Association 1994) or the Clinician-Administered PTSD Scale (CAPS; Blake et al 1995). (For 58 subjects, PTSD diagnosis was determined through administration of the CAPS.

The remaining five subjects were not administered the CAPS. Their PTSD diagnoses were obtained through administration of the SCID.) At testing, subjects ranged in age from 42 to 48 years (mean = 45.2, SD = 3.1). Comorbid diagnoses were common: 84% met criteria for recurrent major depressive disorder (MDD), 60% for current MDD, 74% for history of alcohol abuse/dependence, and 65% for history of illicit substance abuse/dependence. Other psychometric features confirmed the highly symptomatic condition of this sample: mean CAPS total severity was 81 (SD = 17), mean Mississippi Scale (MISS; Keane et al 1988) score was 123 (SD = 15), mean Beck Depression Inventory (BDI; Beck et al 1961) score was 24 (SD = 10), and mean Combat Exposure Scale (CES; Keane et al 1989) score was 27 (SD = 8).

Procedures

All subjects gave informed consent. Subjects slept 3 or 4 nights in the sleep laboratory located immediately adjacent to the inpatient unit. Scheduling was arranged to accommodate subjects' typical bedtimes. Inpatient subjects terminated their sleep at will, but not later than 6:00 AM, the standard wake-up time for the inpatient program. Subjects made entries in a sleep diary before and after sleep, and during episodes of awakening during the night. This diary enabled subjects to indicate recall of dream mentation in the categories "Vietnam nightmare," "unpleasant dream," and "pleasant dream."

The recording montage included two channels of bipolar electro-oculography, four channels of scalp electroencephalography (EEG; F3, F4, Cz, and Pz referred to linked mastoids), mentalis and left anterior tibialis electromyograms, abdominal respiratory effect, electrocardiograms, and blood oxygen saturation. Electrocardiograms were recorded using the "lead 1" derivation and filtered to 1 to 30 Hz before digitization. Electro-oculograms and EEGs were filtered to a 0.3- to 30-Hz bandwidth. Electromyograms were filtered to a 30- to 300-Hz bandwidth, then rectified and integrated over a 20-msec time-constant. After conditioning, all physiologic data were digitized at 125 Hz and streamed to disk. Manual sleep staging of paper records was performed by trained sleep technicians following standard criteria applied to 30-sec epochs. Electroencephalograms from the Cz site were used for sleep staging. Indices of sleep architecture extracted included time asleep; time in stages 1, 2, 3, and 4 and rapid-eye movement (REM) sleep; awake and movement times; and latencies to sleep and REM. Three different REM latencies were computed: 1) latency from the advent of three consecutive epochs of stage 1, 2) latency from the advent of three consecutive epochs of stage 2 sleep, and 3) latency from "stabile sleep." Stabile sleep was defined as beginning with the first epoch at which 10 min of stage 2, 3, or 4 sleep had accumulated, with that accumulation "penalized" at a rate of 1:1 for intervening stage 1, and 2:1 for intervening wake. The per-subject values presented below are, in all cases, means calculated over all postadaptational nights.

Results

In most cases, the following analyses utilize two-factor analyses of variance or multivariate analyses of variance (MANOVAs) in which TRN and NTRN complaint are limited to two levels, presence versus absence, to maximize cell sizes.

Psychometrics

Nightmare complaints were assessed via a sleep history questionnaire (Figure 3). As shown in Table 1, 48 of 63 PTSD patients endorsed TRNs, whereas 39 of 63 endorsed nontraumatic nightmares. The distributions of these endorsements were significantly different [$\chi^2(1) = 6.9$, $p = .009$], as in the larger sample. Analysis of the effects of TRN and NTRN complaint on CAPS total severity scores (from which the nightmare item had been excluded) found no significant effects for either nightmare type [TRN, $F(1,54) = 2.4$, ns; NTRN, $F(1,54) = 0.6$, ns] and no interaction [$F(1,54) = 0.01$, ns). Similarly, a MANOVA performed upon CAPS Criterion B (excluding the nightmare item), C, and D severities found no significant omnibus F tests for either nightmare type [TRN, $F(3,52) = 0.8$, ns; NTRN, $F(3,52) = 0.13$, ns] and no interaction [$F(3,52) = 0.4$, ns]. Trauma-related nightmare complaint was associated with a large effect on the CAPS nightmare item itself [$F(1,54) = 16.6$, $p < .001$], whereas NTRN was not [$F(1,54) = 0.87$, ns], and the two factors again did not interact [$F(1,54) = 1.4$, ns]. Insofar as the CAPS nightmare item is intended to assess TRNs, specifically, these findings were not surprising; however, they support the convergent validity of both assessments in this sample, as well as the distinction between TRN and NTRN complaint. Though tangential to the main thrust of this report, it was of interest to analyze effects of TRN complaint on the CAPS sleep initiation/maintenance and startle items. In both cases, the effect of TRN proved significant [sleep maintenance, $F(1,54) = 5.7$, $p = .02$; startle, $F(1,54) = 5.4$, $p < .02$]. The effect of NTRN complaint was not significant and the two factors did not interact. In this sample, nightmare complaint was not associated with significant effects on CES, BDI, or MISS scores or scores on scales 6 or 8 of the MMPI/MMPI-II. Thus, associations between nightmare complaint and both CES and MMPI scales 6 and 8 elevations seen in the larger sample were not replicated here, though trends were in the same directions.

Sleep Architecture

Nightmare complaint was not associated with how long PTSD patients slept in the laboratory (all Fs < 1). Though sleep efficiencies were high, this variable exhibited a nominally significant univariate effect of TRN complaint [$F(1,59) = 3.95$, $p = .05$], with TRNs associated with less efficient sleep (91% vs. 94%). Non–trauma-related nightmare complaint did not show such an effect [$F(1,59) =$

Table 2. Sleep Continuity Measures by Group

	Group (trauma-related nightmares/non–trauma-related nightmares)							
	−/− (N = 10)		−/+ (N = 5)		+/− (N = 14)		+/+ (N = 34)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Minutes asleep	334.7	45.4	337.7	66.9	346.0	45.7	346.1	46.2
Sleep efficiency	92.4	3.2	95.9	2.6	90.7	6.2	91.1	5.4
Sleep latency	5.4	2.5	6.8	6.2	5.8	2.5	6.5	4.5
Wake-after-sleep-onset	21.0	8.5	7.1	9.5	31.2	26.9	28.5	24.2
Movement time	1.9	2.2	3.0	3.3	0.9	1.0	1.5	1.9

Tabulation of sleep continuity indices by nightmare group. Trauma-related nightmare complaint was associated with significant reductions in sleep efficiency that proved specific to wake-after-sleep-onset (see text for details).

1.0, $p = .3$], nor was there an interaction of TRN and NTRN complaint [$F(1,59) = 1.8$, $p = .18$]. When sleep efficiency was decomposed into sleep latency, wake-after-sleep-onset, and movement time, additional differences emerged (Table 2). The omnibus F for TRN complaint was nominally significant [$F(3,57) = 2.7$, $p = .05$], whereas that for NTRN complaint was not [$F(3,57) = 1.3$, ns], nor did the factors interact. Trauma-related nightmare complaint was not associated with a univariate effect on sleep latency [$F(1,59) = 0.002$, ns], but instead with effects on wake-after-sleep-onset [$F(1,59) = 4.9$, $p = .03$] and movement time [$F(1,59) = 4.0$, $p = .049$]. Subjects endorsing TRNs exhibited more wake-after-sleep-onset (29 min vs. 16 min) than those that did not. Expressed as a percentage of sleep, the respective values were 7.5% of sleep versus 4.6% of sleep [$F(1,59) = 5.5$, $p = .023$]. Unexpectedly, subjects with TRN complaint exhibited less movement time (1.3 min vs. 2.0 min) than those denying TRNs. Movement time was nonnormally distributed, and so was reanalyzed after square-root transformation. After transformation, the effect of TRN on movement time was not significant [$F(1,59) = 2.6$, ns].

Table 3 presents the distribution of sleep to stages 1, 2, 3, and 4 and REM over groups. A MANOVA of absolute times in stage did not yield a significant omnibus F test for TRNs [$F(5,55) = 1.2$, ns], NTRNs [$F(5,55) = 0.51$, ns], or their interaction [$F(5,55) = 1.1$, ns]. Because stage 4 sleep had shown a reduction in PTSD patients in at least three prior studies (Glaubman et al 1990; Kramer and Kinney 1985; Woodward et al, in press), the univariate effect of TRN frequency on stage 4 minutes was nevertheless analyzed and found to be significant [$F(1,55) = 6.14$, $p = .015$], with TRNs associated with reduced stage 4 sleep (1.0 min vs. 4.3 min); however, because stage 4 sleep time was nonnormally distributed, and remained nonnormal after various transformations, the effect was retested with the Mann–Whitney U. In this case, the trend toward reduced stage 4 sleep in association with TRN was not statistically significant ($U = 419$, $p = .241$). Because sleep times were comparable and sleep efficiencies high, a parallel analysis of percent times in stages 1–4 and REM produced essentially the same results and will not be further reported.

There were no effects of nightmare complaint on REM latency from stage 1, REM latency from stage 2, or REM latency from stabile sleep (all Fs < 1). Mean values for these measures were 65 min (SD = 29), 63 min (SD = 25), and 49 min (SD = 19), respectively.

Effects of nightmare complaint were tested for attributability to MDD or alcohol dependence. In the interests of brevity, NTRN complaint was eliminated from these analyses. In this analysis, small cell sizes emerged, particularly for subjects without TRNs and absent histories of

Table 3. Sleep Stage Distribution by Group

	Group (trauma-related nightmares/non–trauma-related nightmares)							
	−/− (N = 10)		−/+ (N = 5)		+/− (N = 14)		+/+ (N = 34)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Stage 1 (min)	36.7	13.0	39.6	26.9	42.4	21.3	42.7	27.4
Stage 2 (min)	180.2	40.8	169.1	72.9	194.9	41.6	183.7	37.9
Stage 3 (min)	27.7	24.0	23.9	32.4	18.9	21.1	27.7	23.1
Stage 4 (min)	3.1	7.7	9.7	21.7	1.2	4.1	1.0	3.6
REM (min)	87.1	28.2	95.4	25.1	88.6	19.8	91.0	19.5
Stage 1 (%)	10.9	3.1	11.2	6.6	11.9	5.3	11.9	6.5
Stage 2 (%)	53.7	10.5	49.2	15.2	56.4	9.7	52.9	8.7
Stage 3 (%)	8.6	7.5	8.8	11.8	5.6	6.8	8.4	7.6
Stage 4 (%)	1.1	2.9	3.0	6.8	0.4	1.5	0.3	1.2
REM (%)	25.9	7.4	27.7	5.8	25.6	5.9	26.4	4.6

REM, rapid eye movement.

MDD or of ETOH. As a result, power to test interactions was compromised. The presence or absence of TRNs did not significantly differ as a function of whether PTSD patients met criteria for current MDD [$\chi^2(1) = 1.5$, ns], recurrent MDD [$\chi^2(1) = 0.25$, ns], or ETOH [$\chi^2(1) = 0.66$, ns]. Current MDD exerted no effect on wake-after-sleep-onset [$F(1,59) = 0.11$, ns] and did not interact with TRN complaint [$F(1,59) = 0.002$, ns] to influence wake-after-sleep-onset. Similarly, recurrent MDD (contrasting patients who reported never having had a depressive episode with those who had) exerted no effect on wake-after-sleep-onset [$F(1,59) = 0.009$, ns] and did not interact with TRN complaint [$F(1,59) = 0.07$, ns]. Finally, the effect of ETOH on wake-after-sleep-onset was not significant [$F(1,59) = 1.4$, ns] and did not interact with TRN complaint [$F(1,59) = 0.22$, ns]. Thus, elevation of wake-after-sleep-onset associated with elevated TRN complaint was independent of the principal comorbid diagnoses accompanying PTSD.

Insofar as TRN complaint was associated with both increased wake-after-sleep-onset and increased severity ratings on the CAPS sleep maintenance item, it was of interest to test the relationship between the latter two variables. In this sample, a significant positive correlation was observed between wake-after-sleep-onset in the laboratory and CAPS sleep maintenance severity ratings ($\rho = .28$, $p = .039$).

Discussion

In this sample of chronic, severe, combat-related PTSD patients studied in the laboratory, TRN complaint was associated with increased wake-after-sleep-onset, whereas NTRN complaint was not. The two classes of nightmare complaint did not interact to influence wake time. Increased wake-after-sleep-onset was not attributable to comorbid MDD or ETOH. The increase of wake-after-sleep-onset in patients endorsing TRN complaint was approximately 13 min per night, a percentage increase of 81%. It is not clear whether this increase should be considered clinically significant. An important obstacle to making such a determination is our ignorance of how observations made under the "guarded" conditions of the sleep laboratory translate to habitual sleep in PTSD patients. The high sleep efficiencies observed and the absence of adaptation effects reported previously in our laboratory (Woodward et al 1996a) are consistent with morning reports from many of our subjects that they slept better in the lab than under normal conditions. It must be emphasized that the observed association between nightmare complaint and wake-after-sleep-onset is purely correlational. Arguing from these data, one could posit that nightmares result in a tendency to arouse from sleep, or that a tendency to arouse from sleep leads to reports of anxiogenic dream mentation (Schredl et al 1998).

These results share common features with those of Kramer and Kinney (1988), who compared the sleep of eight combat veterans with laboratory-verified TRNs to that of eight combat veterans with some features of PTSD but without TRNs. They observed no overall differences in sleep efficiency between groups, and no effect on sleep latency, but they did observe, on the last (fourth) night of the study when subjects were presumably well adapted to the laboratory, that subjects with TRNs woke up more often than subjects without. Although Kramer and Kinney also observed delayed REM sleep onset in subjects with nightmares, this study found no effects on REM sleep architecture. In general, REM sleep amounts were mildly elevated and REM latencies were at the low end of the nonpathologic range (Kupfer et al 1986). Findings with regard to REM sleep have been notably varied in PTSD (Ross et al 1989). Among the 12 studies referenced in the Introduction, laboratory-observed nightmares were as likely to appear in association with non-REM (particularly stage 2) sleep as with REM sleep. If TRNs are not fundamentally REM sleep phenomena, then perhaps frank modifications of REM sleep amounts and/or timing should not be expected. In fact, the current state of the literature is such that one might with equal justification posit REM nightmares to be associated with REM "disruption" (Mellman et al 1995b), as with "REM pressure" (Ross et al 1994). Understanding specific effects of nightmares on REM sleep architecture may remain impossible without further direct polysomnographic observations of these events.

The observation that TRN complaint was associated with effects on objective sleep whereas NTRN complaint was not recalled their psychometric divergence in the larger sample of similarly classified veteran inpatient subjects. Although associations between TRN and combat exposure and MMPI/MMPI-II subscale scores were not replicated in the sleep laboratory sample, the laboratory sample evidenced a familiar pattern of association between nightmare complaints and the CAPS nightmare item: TRN complaint, but not NTRN, corresponded to elevations on the CAPS nightmare item, and the two factors did not interact. Taken together, these findings suggest that for PTSD patients TRNs are distinct from non–trauma-related unpleasant dreams not only in their phenomenology, but also in their associations with memories of trauma, with other domains of psychologic function, and with sleep behavior.

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